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CONTRACTING ORGANIZATION: Baylor College of Medicine
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14. ABSTRACT

The goal of the project is to provide summer research experiences in prostate cancer and a series of ancillary activities to 6 students with the goal of interesting them in careers in prostate cancer and to prepare them for graduate school/medical and subsequent careers in prostate cancer. We successfully recruited two students from PVAMU and four from XULA for the summer research program. Students participated in research experiences, seminars, workshops, and supplementary activities. Outgoing interviews confirmed that the program was a valuable experience for the students and that the students would recommend the program to other students.

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Table of Contents

Page

1. INTRODUCTION. This project builds on and extends an 8-year partnership between Prairie View A & M University (PVAMU) and Baylor College of Medicine (BCM) thorough adding Xavier University of Louisiana (XULA) as an HBCU partner. Our goal is to encourage and begin to prepare undergraduates to enter careers in prostate cancer research. This project will provide a 9-week summer mentored prostate cancer research experience at BCM for a total of 6 undergraduates/year from PVAMU and XULA. Students will work at the forefront of research, often using high tech equipment typically not available to undergraduates. Participants attend weekly research discussion group/seminars focused on prostate cancer, which will provide opportunities to become better acquainted with prostate cancer researchers from Ph.D. students to faculty as well as various areas of prostate cancer research. They will make PowerPoint presentations and submit critiqued written summaries (abstracts) of their work at the end of the program. Most prepare posters to present at research conferences. The participants will be co-registered in the SMART summer undergraduate research program at BCM. This program includes unique daily interdisciplinary seminar series, career development activities like GRE Prep and graduate school application workshops and career counseling.

2. KEYWORDS: HBCU, students, training, research, seminars

3. ACCOMPLISHMENTS

Major goals and accomplishments

Major Task 1 Advertising and Recruiting. At PVAMU, Dr. Regisford advertised the program and arranged for Drs. Weigel and Dr. Slaughter to present the program to interested students and to answer questions about the program. She also assisted the students in preparing applications. Dr. Payton-Stewart and Dr. Thomas advertised the program at XULA, prepared announcements and hosted Drs. Weigel and Slaughter during their visit. Drs. Weigel and Slaughter spoke to a large group of interested students.

Major Task 2 Screening applicants/acceptance/ and assignment to labs. Drs. Regisford and Payton-Stewart spoke to candidate students at their institutions and provided feedback to BCM. Drs. Weigel and Slaughter reviewed the candidates and picked the most promising candidates from the two schools. In this year, we had many more highly qualified candidates from XULA than from PVAMU resulting in our selecting 4 from XULA and 2 from PVAMU for the program. Dr. Weigel surveyed candidate mentors and matched mentors with the candidates.

Major Task 3 Summer program. Each of the six students participated in a 9 week research program. All participated in the specialized prostate cancer seminars and many, if not all, of the noon seminar programs. Several took the GRE Prep course and the supplementary training specifically for students in this program. Some found it extremely valuable. Others, who were more oriented to medical school were somewhat less interested although we modified the supplementary material this year to more directly pertain to both graduate and medical school exam preparation. Students also participated in other activities including a welcome social to meet other prostate cancer faculty beyond their mentors and met with peer mentors who helped them in adjusting to BCM and answered questions about research and graduate school. The students found this to be a very useful aspect of the program. All students wrote abstracts (see abstracts under Products), prepared and gave oral presentations on their work. At least one student will be presenting her work at ABRCMs. Dr. Weigel conducted exit surveys and final evaluations were collected from mentors. The students were very positive about the program. All had very positive experiences in the laboratories and found the seminars and other programs useful. Some were interested in additional social activities, while others were not.

Major Task 4 Extended mentoring. Students have discussed their experiences with their HBCU advisors both by email and in person. As planned this task was initiated subsequent to the completion of the summer program. Trainees contact mentors for preparation of posters for local and national presentations. We have a small amount of money remaining and have been granted a no cost extension. Thus, recruiting for the final slots will be done at PVAMU and XULA by the local mentors with help from former program members rather than

spending money for travel. Some of the trainees will be presenting their BCM work at local research days and at ABRCAMS. At least one student will be presenting her work at ABRCAMs.

Opportunities for training and development. This is a training grant, so there were many opportunities for training and development. All students participated in mentored research programs, general seminar programs, prostate cancer seminar programs, most in a GRE prep course and all prepared and presented research presentations.

Dissemination of results: /A

Plans for the next reporting period. This was the final year of full funding. Recruited students for next year will participate in all planned activities of the previous years with the minor exception of the additional supplementary workshops, which are quite expensive to present..

4. IMPACT:

Principal discipline(s) 6 students were introduced to prostate cancer research and related areas of research
Other disciplines: N/A
Technology transfer: N/A
Impact on Society: Students will bring their expertise in prostate cancer back to their schools and communities.

5. CHANGES/PROBLEMS: No significant problems, so no significant changes are planned.

6. PRODUCTS: Abstracts of oral presentations at BCM

The Therapeutic Effects That Ginseng Compounds Have on Prostate Cancer Cells Jessica Carmon Mentor: Li Xin

Ginseng is a species of plants with fleshy roots that have been used in traditional medicine for many centuries. However, modern research is inconclusive about the biological effects of ginseng, thus the purpose of my project was to determine the effects that ginseng compounds have on the viability prostate cells. I tested 4 different cell lines for my experiment: WPMY-1, 22RV-1, C4-2, and LNCaP. I did this to determine whether the effects of ginseng are similar across each cell line. I hypothesized that ginseng compounds would decrease the viability of cancerous cell lines, but increase or have no effect on the noncancerous cell line, WPMY-1.

Previous studies have shown that the active constituents found in ginseng have cytotoxic and anti-inflammatory effects. In the prostate, inflammation plays a critical role in the initiation and progression of prostate cancer as well as benign prostatic hyperplasia. Benign prostatic hyperplasia, or BPH, is basically just an enlarged prostate gland that is neither cancerous nor life threatening, however, it can lead to serious problems as it develops over time. It’s important to take note that it’s the inflammation that causes BPH to become cancerous. Therefore, if we can prevent inflammation of the prostate gland, then we can prevent the initiation and progression of prostate cancer.

In order to test my hypothesis, I split up 4 different cell lines onto 4 different 96-well plates, and put approximately 4000 cells per mL in each well. I set the plate up in triplicates, so there were 3 wells for each compound and each concentration. Next, I had to dilute the ginseng compounds that were used, but because some of these compounds are nonpolar, they had to be dissolved in ethanol instead of water. In case the ethanol had an effect on the cell, I set up one control of cells with no ethanol or no added compound, and I also set up another control that contained cells with just ethanol. Next, I diluted the compounds to 1 milli-Molar, and added a certain amount of each compound to each well depending on the concentration of compound that I was adding. Then, I waited 48 hours to count the cells with a hemocytometer, and then I compared the results on an Excel spreadsheet.

What I found was that the ginseng extracts did in fact decrease the cell viability of prostate cancer cells, however most of them also decreased the cell viability of the noncancerous cell line as well. If I had more time to continue this project I would continue to test all of my compounds and determine why the compounds killed certain cell lines but did not kill others. For example, what chemical property of a certain compound contributed to the ginseng being able to kill a cell line, that another compound was missing to be unable to kill the cells. Overall, ginseng compounds decreased the viability of prostate cancer cells, which means that ginseng could possibly inhibit the initiation and progression of prostate cancer.

Targeted Degradation of HIV-1 Protease Utilizing Ubiquitin-Proteasome Pathway

Do, Camilla, Xavier University of Louisiana, Class of 2019
Laboratory of Michael A. Mancini, Ph.D. (IMC) and Damian Young, Ph.D.
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Targeted degradation of proteins can offer a new means of treatment for several human maladies. PROTACs, or Proteolysis Targeting Chimeras are small molecules designed to selectively induce degradation of target proteins. Their bifunctional structure brings together a protein of interest to an E3 ligase enzyme, hijacking the Ubiquitin-Proteasome System (UPS). PROTACs have been shown to successfully promote degradation of several cancer-related proteins, including the androgen receptor, which is dysregulated in certain prostate cancers.

Overall, the goal of my project was to aid in the development of a platform utilizing known drugs as starting points for PROTAC design. For such, a viral infection system was selected as proof-of-concept platform because of the wide availability of small molecule drugs, cellular reporter assays and clinical relevance.

Working with Wanderson Rezende, our research focused on the development of a novel PROTAC for targeted degradation of HIV-1 protease (HIV-1 PR). Human immunodeficiency virus type 1, or HIV-1, is a retrovirus that require an aspartyl protease to cleave polypeptide chains into individual functional proteins, and in the process, allowing the virus to mature and continue to replicate. Therefore, it is hypothesized that by targeting HIV-1 PR for degradation, the virus replication cycle will be interrupted. Our PROTAC compound will recruit HIV-1 PR through a protease inhibitor moiety, and a small molecule moiety to recruit the E3-ligase, both moieties connected through a flexible linker. The chosen protease inhibitor moiety is Amprenavir, an FDA approved drug for HIV-1 treatment. The linker, a long carbon chain, will promote protein-protein interaction between HIV-1 PR and E3 ligase cereblon. Pomalidomide will bind to the E3 ligase which in turn will recruit the remaining UPS machinery to promote polyubiquitination of HIV-1 PR.

The laboratory of Dr. Damian Young is focused on fragment-based drug discovery, synthetic organic chemistry, and diverse-oriented synthesis. Dr. Michael Mancini's laboratory is part of the Integrated Microscopy Core, and with his co-mentorship and facility, I performed multiple fluorescence assays and acquired multiple microscopy images throughout the summer with the goal of characterizing key steps of the design of a PROTAC against HIV-1 PR.

At each synthetic step of the PROTAC, the Amprenavir-based intermediates were tested in cells using a fluorescence reporter assay to evaluate if the protease inhibitor moiety was in fact binding to HIV-1 PR. A plasmid encoding for a fusion protein GFP-HIV PR was transfected in A549 and HEK293 cells and further treated with varying drug concentrations. Higher concentrations of protease inhibitors correlated with more intense GFP signals. Compounds and intermediates include the Amprenavir, fluoro-amprenavir, and linker-attached amprenavir. After imaging the cells for GFP intensities, cells were labeled with DAPI (nuclear stain) and Phalloidin (cytoskeleton stain). This allowed us to physically visualize transfection efficiency and GFP intensities. Various experiments were performed in attempts to optimize our assay, such as the seventy-two-hour

live imaging and different treatment times of the drugs. We established that optimal reading times for the assay are between 24-36h post-treatment with drugs and that all compounds tested bind to the HIV-1 PR at a dose-dependent manner.
To test for the presence of the E3-ligase cereblon in our cell lines, A549 and HEK293, immunoblotting and immunofluorescent labeling were done. Cells cultured on coverslips were immunofluorescent labeled with anti-CRBN, but signals were weak even at high antibody concentrations such as 4ug/mL. Immunoblots were also done, but only nonspecific bands were obtained with the antibody tested. In addition, a transfection plate was also labeled with anti-HIV PR. We were able to see the expected co-localization of HIV-1 PR and GFP, confirming the presence of the GFP-HIV-1 PR fusion protein in the transfected cell lines.
Future directions include finishing the synthesis of the PROTAC, improving its synthesis yield, and testing the PROTAC in cells. Assays for the PROTAC's effectiveness in promoting HIV-1 PR degradation in transfected cells should also be done. Labeling and immunoblotting for anti-GFP and anti-HIV PR in cells treated with the PROTAC would be a good comparison with cells treated with just protease inhibitors. If PROTAC-mediated degradation of HIV-1 PR occur, no GFP signals or HIV-1 PR signals will be identified by either immunoblotting or immunofluorescence labeling. Lastly, testing for different linkers in the PROTAC construction should be done to find the optimum linker distance that promotes the most protein-protein interaction and degradation of HIV-1 PR.

Characterizing the Stability of PRMT5 in Various Conditions

Martin, Karla, Xavier University of Louisiana, Class of 2019
Laboratory of Dr. Damian Young, Ph.D.
Department of Pathology and Immunology, Department of Pharmacology, Center for Drug Discovery, Baylor College of Medicine

Dr. Damian Young's laboratory in the Center for Drug Discovery is focused on developing a fragment-based library for screening protein drug targets. Protein arginine methyltransferase 5(PRMT5) has been identified as a possible drug target for drug discovery. Recent literature has shown 15% of cancer cases are sensitive to its inhibition (Beroukhim et al., 2010). This PRMT5 dependence is specifically present in cancer cell lines with Methylthioadenosine phosphorylase (MTAP) deleted as a consequence of tumor suppressor deletion. The tumor suppressor, p16/CDKN2A gene is located on chromosome 9, in close proximity to MTAP. The frequency of cancer cells missing copies of both CDKN2A and MTAP is approximately five percent in prostate cancer. Additionally, in prostate cancer, PRTM5 interacts with the transcription factor, ERG, to suppress genes that prevent prostate cancer metastasis. Considering no drug or therapy currently exists to target, ERG directly, PRMT5 can potentially function as a lethal drug target to repress prostate cancer proliferation since the two co-exist in a complex (Mounir et al.,2016).

My research goal this summer was to determine stable conditions for PRMT5. To do this, I employed two assays, the thermal shift assay to evaluate PRMT5 stability using melting temperature data and the methyltransferase activity assay to assess the activity of PRMT5. My first task involved the use of the thermal shift assay to narrow down possible buffer additives that contribute to the protein's stability for use in future assays. This assay involved testing sixteen additives against PRMT5 and calculating the melting temperature(T_m) for the protein under a specific additive condition to determine if the additive destabilized or stabilized the protein. The results of the initial screen, revealed that PRMT5 was stabilized by glycerol, calcium chloride, and S-adenosyl-L-methionine (SAM). From this data, a calcium chloride dose response test was performed to examine whether varying doses of calcium chloride effect PRMT5 stability. The results from the assay suggested that the stability of PRMT5 is not dose dependent. From this preliminary data, we continued

testing calcium chloride independently and in conjunction with known binders, however the same stabilizing effect of PRMT5 was not observed. This inconsistency in the assay data lead to the use of the methyltransferase activity assay to test the activity variation between different samples of recombinant PRMT5 that have been used for the assays. The assay results showed a substantial difference in activity between the four batches of PRMT5 used. With this information, it appears that there may be a correlation between the activity of PRMT5 and its ability to be stabilized by calcium chloride. With this information, we decided to run future assays with the most active samples of PRMT5.

We will continue to characterize the stability of PRMT5 in the presence of calcium chloride to better understand the stabilizing effect of calcium chloride on the recombinant protein as a buffer additive. The extent to which PRMT5 regulates biological pathways will also be explored to better understand the degree to which PRMT5 knockdown effects pathways linked to certain cancers and cardiovascular diseases. After complete characterization of PRMT5 and assay optimization, commercial and in-house fragments will be screened against PRMT5 for fragment based drug discovery.

Role of *KCTD13* in the Prostate and Its Potential Impact in Prostate Cancer

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The focuses of Dr. Dolores Lamb's laboratory are the mechanisms controlling male reproductive function and genitourinary (GU) diseases caused by genomic disruptions, specifically microdeletions and microduplications. Previously the lab identified a hotspot on chromosome 16p11.2 was found to be associated with GU defects. A patient with a small microdeletion within that region revealed several candidate genes for impacting genitourinary tract function, one of which is the dosage sensitive gene *KCTD13.* KCTD13 is a part of a E3 ubiquitin-protein ligase complex and influences androgen receptor (AR) signaling. For my project, I was assigned to gather preliminary data on the potential role of *KCTD13* in normal and malignant prostate cells.

The growth of prostate cancer is influenced by a category of steroid hormones called androgens. Testosterone, a form of androgen, is secreted by the testis and adrenal glands. Upon entering the prostate testosterone is metabolized to dihydrotestosterone (DHT), which then binds and activates androgen receptor (AR). The AR complex translocates to the nucleus and activates genes involved in cell growth. Therefore, my first goal for this project was to determine the effect of *KCTD13* knockdown (KD) and overexpression on AR signaling in both mouse and human prostate cells. I transfected the mouse and human cell lines with siRNA targeting *KCTD13* and a plasmid that overexpresses *KCTD13*. After the transfection, I collected the cells and extracted the RNA. After I quantified the RNA, I analyzed the mRNA expression levels of the target gene via RTPCR. In both the human and mouse cell lines we were able to successfully knockdown and overexpressed *KCTD13.* The next step is to analyze any changes in expression levels AR downstream targets.

My second goal, was to evaluate the prostate morphology in wildtype (WT) and *Kctd13* knockout (KO) mouse prostates. I microdissected prostates from both WT and KO mice. Once the prostate was isolated from the genitourinary tract, it was fixed and put in paraffin blocks. Then I sectioned the blocks and performed a Hematoxylin and Eosin staining. In the KO mice, the ventral lobe had a more columnar appearance compared to that of the WT mouse prostate. The prostate epithelial cells are responsive to androgen, so we hypothesized that by removing *Kctd13*, AR signaling is disrupted resulting in less cell proliferation consistent with other phenotypic changes in the *Kctd13* null mice. The results suggested that *KCTD13* is involved in androgen signaling in human and mouse prostate and that the knockdown of the gene leads to less androgen receptor

translocation to the nucleus causing decreased cell proliferation. Our next steps would be to evaluate AR downstream signaling changes in response to *KCTD13* alterations in human and mouse cell lines as well as assessing AR levels in WT and KO mice prostates via immunohistochemistry staining. The results may identify a previously unrecognized pathway impacting prostate growth.

Models for Cancer Immunotherapy

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One of the primary objectives of the laboratory of Dr. Levitt and Dr. Decker is using canine tumor models to develop immunotherapies for human disease. The canine tumor shares specific characteristics with humans such as cellular heterogeneity, expression of checkpoint ligands, immune cell infiltrates and reactive stroma. Humans express genes for MHC classes I and II on chromosome 6. MHC class I is found on all cells. It presents self-antigens and through cross presentation express foreign antigens. MHC class 2 is present on antigen presenting cells like dendritic cells. The aims of this experiment conducted by Vanaja Konduri Ph.D. are: 1) to determine the mutational load and Dog Leukocyte Antigen class 1 binding neo-antigens in canine tumors. 2) Validate predicted DLA class I binding neo-antigens and determine the immunogenicity of validated canine tumor neo-antigens. I worked on isolating DNA/RNA using the FFPE DNA/RNA isolating kit with the help of Vanaja Ph.D. and Jonathan Vasquez. The samples we generated was sent for quality control. These samples were then used for library prep and the RNA was sent for exome sequencing. I worked on amplifying plasmid DNA by performing several mini preps and growing HEK 293 cells. The cells would be transfected with the plasmid DNA to construct the DLA chimera. The mini preps I performed helped in the identification of unique DLA alleles. My summer research project was focused on studying tumor growth *in vivo*. I assisted Vivian with this project. Vivian is the graduate student in the lab. She had developed a cell line for pure BL6 mice. The plasmid contained luciferase and a neomycin drug selection. I grew and split the cells. This project was focused on investigating tumor growth in different models. They are: orthotopic, ectopic and metastatic models. I inoculated a quarter million cells into each mouse through different methods. I inoculated mice subcutaneously and intraperitoneally while Vivian performed the intravenous injection. We observed the growth of the tumor by imaging. The In Vivo Imaging System (IVIS) at TMF was used to generate images at three different time points. There was an improved growth rate compared to the last cell line that was used. Developing dendritic cell vaccines to inhibit tumor growth is the next step to tackle prostate and other cancers.

Characterizing the Behavior of Androgen Receptor Splice Variants in Androgen Dependent Prostate Cancer Cells

Turner, Williamson D., Xavier University of Louisiana, Class of 2018 Nancy Weigel Ph.D., Department of Molecular Cell Biology, Dan L. Duncan Cancer Center, Baylor College of Medicine

In prostate cancer, metastatic tumor growth is driven by the androgen receptor (AR). A common mechanism to combat such growth is androgen ablation therapy (chemical castration). The therapy removes circulating androgens from the bloodstream to cut off the supply of androgen to the AR. Initially, this type of therapy successfully slows metastatic tumor growth. However, after some of the treatment is completed, metastatic growth begins to redevelop independent of circulating androgens. The primary interest of Dr. Weigel's lab is to define the mechanism by which the AR circumvents its reliance on circulating androgen. She currently

hypothesizes that one method that may result in androgen independence might be alternate splice variants of the AR gene. Not only are these variants active in the absence of androgen, but gene expression studies suggest that they may also regulate gene expression to induce SRD5A1 which catalyzes the conversion of androstenedione (AD) into dihydrotestosterone (DHT) through a bypass pathway, and to repress UGT2B15 and 17 which catalyze the degradation of DHT. The goal of my research this summer was to identify a possible test gene with which to investigate the ability of two AR splice variants (ARV7 and ARV567) to metabolize AD into DHT. To do this, I screened 8 candidate genes that had previously been identified via RNA Seq. in the lab as genes that may be induced by the full length receptor but not in the variants. To test these genes, I harvested LNCaP cells that expressed full length receptor, ARV7, and ARV567 respectively. I induced the expression of the variants by adding doxycycline and then extracted RNA from each cell type and used it do qPCR for each of the 8 genes. Of those 8 genes, I was able to identify one (TMEM100) that is induced by the full length receptor but not induced by either variant. Using the TMEM100 gene, the lab will be able to perform experiments to test the AD to DHT metabolic activity of both splice variants. In addition to the gene screen, I also attempted a proliferation assay to analyze the effects of ARV567 on growth rate. However, there is a discrepancy in the growth pattern which requires the conditions to be adjusted before proliferation can be properly analyzed.

7. Participants and other collaborating organizations

Nancy Weigel, PI, no change. No change during the funding period and nobody contributed a full month of effort. For the no cost extension, Dr. Laurie Connor, who already plays a major role in the training, will assume the role of Dr. Gayle Slaughter, since Dr. Slaughter has retired

Active other support: PI. Dr. Weigel's other support increased during the funding period, but, since this is a training grant, there is no overlap and she had ample % effort remaining to carry out the obligations to this training grant. The new funding during this period includes a developmental project on the MD Anderson Cancer Center Prostate Cancer SPORE, P50 CA140388A1 Logothetis (PI), W81XWH-17-1-0236 Weigel (PI) (2.4 months), R01 CA207270 (Fuqua) Weigel investigator (0.6 months).

Partner organizations: PVAMU and XULA, no change